13 **Samejima H**, Omiya K, Uno M, *et al*. Relationship between impaired chronotropic response, cardiac output during exercise, and exercise tolerance in patients with chronic heart failure. *Jpn Heart J* 2003;**44**:515–25.
14 **Clark AL**, Coats AJ. Chronotropic incompetence in chronic heart failure. *Int J Cardiol* 1995;**49**:225–31.
15 **Robbins M**, Francis G, Pashkow FJ, *et al*. Ventilatory and heart rate responses to exercise: better predictors of heart failure mortality than peak oxygen consumption. *Circulation* 1999;**100**:2411–7.
16 **Beaver WL**, Wasserman K, Whipp BJ. A new method for detecting the anaerobic threshold by gas exchange. *J Appl Physiol* 1986;**60**:2020–7.
17 **Witte KKA**, Clark AL. Is the elevated slope relating ventilation to carbon dioxide production in chronic heart failure a consequence of slow metabolic gas kinetics? *Eur J Heart Fail* 2002;**4**:469–72.
18 **Astrand I**. Aerobic work capacity in men and women with special reference to age. *Acta Physiol Scand* 1960;**49**(suppl 169):1–92.
19 **Katritsis D**, Camm AJ. Chronotropic incompetence: a proposal for definition and diagnosis. *Br Heart J* 1993;**70**:400–2.
20 **Witte KKA**, Thackray SDR, Nikitin NP, *et al*. The effects of α- and β-blockade on ventilatory responses to exercise in chronic heart failure. *Heart* 2003;**89**:1169–73.
21 **Witte KKA**, Thackray S, Nikitin NP, *et al*. The effects of long-term β-blockade on the ventilatory responses to exercise in chronic heart failure. *Eur J Heart Fail* 2005;**7**:612–7.
22 **Lauer MS**, Francis GS, Okin PM, *et al*. Impaired chronotropic response to exercise stress testing as a predictor of mortality. *JAMA* 1999;**281**:524–9.
23 **Lauer MS**, Mehta R, Pashkow FJ, *et al*. Association of chronotropic incompetence with echocardiographic ischemia and prognosis. *J Am Coll Cardiol* 1998;**32**:1280–6.
24 **Lauer MS**, Okin PM, Larson MG, *et al*. Impaired heart rate response to graded exercise: prognostic implications of chronotropic incompetence in the Framingham heart study. *Circulation* 1996;**93**:1520–6.
25 **Endo A**, Kinugawa T, Ogino K, *et al*. Cardiac and plasma catecholamine responses to exercise in patients with type 2 diabetes: prognostic implications for cardiac-cerebrovascular events. *Am J Med Sci* 2000;**320**:24–30.
26 **Dresing TJ**, Blackstone EH, Pashkow FJ, *et al*. Usefulness of impaired chronotropic response to exercise as a predictor of mortality, independent of the severity of coronary artery disease. *Am J Cardiol* 2000;**86**:602–9.
27 **Elhendy A**, Mahoney DW, Khandheria BK, *et al*. Prognostic significance of impairment of heart rate response to exercise: impact of left ventricular function and myocardial ischemia. *J Am Coll Cardiol* 2003;**42**:823–30.
28 **Fei L**, Keeling PJ, Sadoul N, *et al*. Decreased heart rate variability in patients with congestive heart failure and chronotropic incompetence. *Pacing Clin Electrophysiol* 1996;**19**:477–83.
29 **Roche F**, Pichot V, Da Costa A, *et al*. Chronotropic incompetence response to exercise in congestive heart failure, relationship with the cardiac autonomic status. *Clin Physiol* 2001;**21**:335–42.
30 **Azarbal B**, Hayes SW, Lewin HC, *et al*. The incremental prognostic value of percentage of heart rate reserve achieved over myocardial perfusion single-photon emission computed tomography in the prediction of cardiac death and all-cause mortality: superiority over 85% of maximal age-predicted heart rate. *J Am Coll Cardiol* 2004;**44**:423–30.
31 **Keteyian SJ**, Brawner CA, Schairer JR, *et al*. Effects of exercise training on chronotropic incompetence in patients with heart failure. *Am Heart J* 1999;**138**:233–40.
32 **Shakar SF**, Lowes BD, Lindenfeld J, *et al*. Peak oxygen consumption and outcome in heart failure patients chronically treated with beta-blockers. *J Card Fail* 2004;**10**:15–20.
33 **Lechat P**, Hulot JS, Escolano S, *et al*. Heart rate and cardiac rhythm relationships with bisoprolol benefit in chronic heart failure in CIBIS II trial. *Circulation* 2001;**103**:1428–33.
34 **Poole-Wilson PA**, Swedberg K, Cleland JG, *et al*. Carvedilol Or Metoprolol European Trial Investigators. Comparison of carvedilol and metoprolol on clinical outcomes in patients with chronic heart failure in the carvedilol or metoprolol european trial (COMET): randomised controlled trial, *Lancet* 2003;**362**:7–13.
35 **Kinugawa T**, Ogino K, Osaki S, *et al*. Prognostic significance of exercise plasma noradrenaline levels for cardiac death in patients with mild heart failure. *Circ J* 2002;**66**:261–6.
36 **Mehta PA**, McDonagh S, Poole-Wilson PA, *et al*. Heart failure in a district general hospital: are target doses of beta-blockers realistic? *QJM* 2004;**97**:133–9.
37 **Arnold RH**, Kotlyar E, Hayward C, *et al*. Relation between heart rate, heart rhythm, and reverse left ventricular remodelling in response to carvedilol in patients with chronic heart failure: a single centre, observational study. *Heart* 2003;**89**:293–8.
38 **DiFrancesco D**, Camm JA. Heart rate lowering by specific and selective I(f) current inhibition with ivabradine: a new therapeutic perspective in cardiovascular disease. *Drugs* 2004;**64**:1757–65.

IMAGES IN CARDIOLOGY

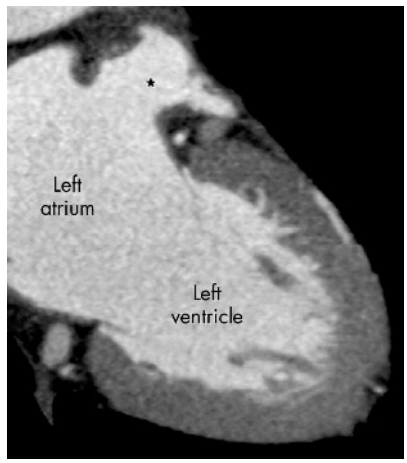
doi: 10.1136/hrt.2005.071704

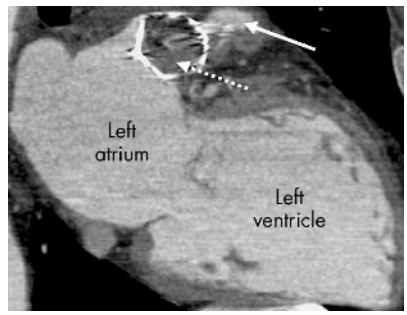
Computed tomography in a patient after percutaneous left atrial appendage transcatheter occlusion (PLAATO)

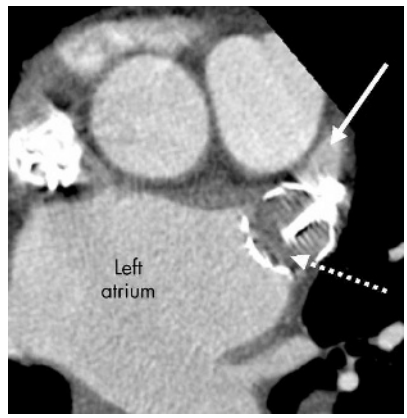
A 61 year-old man with persisting non-rheumatic atrial fibrillation suffered from bleeding due to variable international normalised ratios despite physician guided warfarin treatment. However, without anticoagulation the risk for embolic events would have increased dramatically. The left atrial appendage remained the main location for left atrial thrombus formation, possibly related to the phenomenon of atrial stunning. Therefore, the patient underwent a percutaneous left atrial appendage transcatheter occlusion (PLAATO).

Contrast enhanced computed tomography (CT) (16 row multislice Lightspeed Pro, General Electric) was performed to assess correct placement of the device, to exclude pericardial effusion, and to evaluate possible residual perfusion of the left atrial appendage. The left hand panel shows a non-occluded left atrial appendage (asterisk).

The right hand panels show the correctly sized and placed occluder system (dotted arrow). Despite the presence of atrial fibrillation, pericardial effusion could be reliably excluded. Whereas the occluder system itself is thrombosed, the apex of the left atrial appendage beyond the device is still contrast enhanced (arrow). This finding suggested residual perfusion most likely caused by incomplete coverage with neoendothelial-like cells at the atrial facing surface of the device. Thus the patient received further oral anticoagulation (aspirin) but did not need further warfarin treatment.







O K Mohrs, D Ruebesam, J Peters
o.mohrs@gmx.de